ARTERIOVENOUS MALFORMATION CAUSING HEMORRHAGIC STROKE, TREATED BY RADIOSURGERY USING GAMMA-KNIFE TECHNIQUE

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ABSTRACT

Arteriovenous malformation is a frequent cause of hemorrhagic stroke in young adults and children, with not so many therapeutic options. We present the case of a 26-year-old woman who suffered from a hemorrhagic stroke from an arteriovenous malformation. She underwent radiosurgery three years after the acute event. Two years later, the malformation is excluded from the cerebral circulation.

Key words: arteriovenous malformation, radiosurgery, gamma-knife, hemorrhagic stroke

INTRODUCTION

Arteriovenous malformations (AVMs) are congenital lesions consisting of a tangled web of arteries and veins connected by one or more fistulae resulting in pathological arteriovenous shunting. The vascular conglomerate is called the nidus: it has no capillary bed, and the feeding arteries drain directly to the draining veins. How the abnormal vessels appear or exactly when the process begins is unknown. Most AVMs are located near the surface of the cerebral hemispheres. They tend to enlarge over time and may become calcified. AVMs produce neurological dysfunction through 3 main mechanisms. First, hemorrhage may occur in the subarachnoid space, the intraventricular space or, most commonly, the brain parenchyma: 38-70% of brain AVMs present initially with hemorrhages and hemorrhage from cerebral AVMs represents 2% of all hemorrhagic strokes. Second, in the absence of hemorrhage, seizures may occur as a consequence of AVM: approximately 15-40% of patients present with seizure disorder. Finally, but rarely, a progressive neurological deficit may occur in 6-12% of patients over a few months to several years. These slowly progressive neurological deficits are thought to relate to siphoning of blood flow away from adjacent brain tissue (the "steal phenomenon"). Neurological deficits may be explained alternatively by the mass effect of an enlarging AVM or venous hypertension in the draining veins.

The overall risk of intracerebral hemorrhage from an AVM is of 2-4% per year. However, the prognosis after AVM hemorrhage is generally better than that after intracerebral hemorrhage from other causes; this may be due to the relatively younger age of patients and a greater potential for reorganization of brain function.

Treatment planning for AVMs depends on risk of subsequent hemorrhage, which is determined by

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the demographic, historical, and angiographic features of the individual patient. Treatment methods include medical care, such as anticonvulsant therapy and headache management, and surgical care, be it surgical resection, endovascular embolization or radiosurgery, alone or in any combination. Prior hemorrhage, smaller AVM size, deep venous drainage, and relatively high arterial feeding pressures

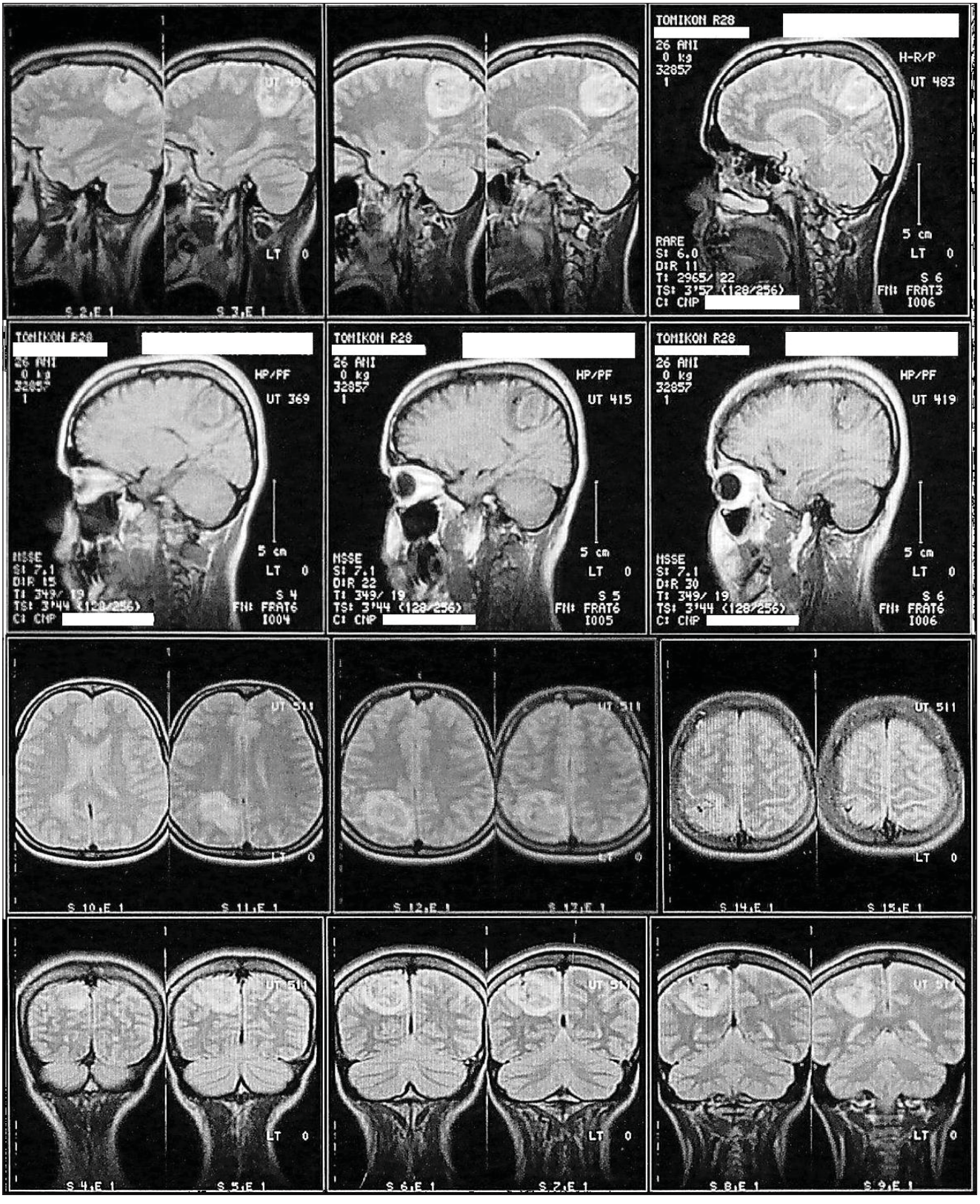

FIGURE 1. *Cerebral MRI showing right parietal hematoma.*

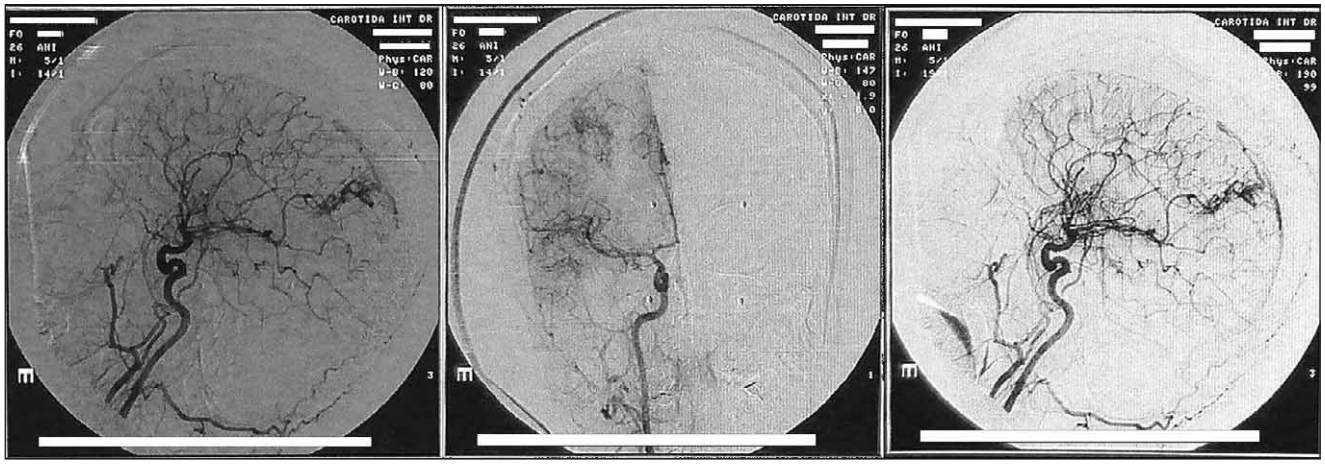

FIGURE 2. *Angiography images of the parietal AVM*

make subsequent hemorrhage more likely. Thus, treatment of AVMs is best achieved with a multispecialty team comprising a neurologist, neuropsychologist, neurosurgeon, interventional neuroradiologist, and neuroanesthesiologist.

CASE PRESENTATION

We present the case of a 26-year-old Caucasian woman first admitted to the hospital in May 2004 with right parieto-occipital headache and loss of muscular strength in the left limbs, which had occurred abruptly during an onstage piano performance, a week before admission.

On hospital admission the patient was alert, the clinical examination revealed no fever, a blood pressure value of 170/100 mmHg, rhythmic heart beats, left hemineglect and left hemiparesis, predominantly brachial. The laboratory tests were in normal range.

An emergency cerebral CT scan followed by a cerebral MRI (Fig.1) showed a cerebral hematoma undergoing resorption, situated in the right parietal lobe, without mass effect on the median structures. An angiographic examination of the cerebral arteries was performed. (Fig.2) It revealed a right parietal AVM with arterial supply from the right posterior parietal artery, nidus dimensions of approximately 2cm (Spetzler-Martin grade I) and superficial venous drainage through a corticalized dilated vein to the superior sagittal sinus.

The symptoms subsided after the first 3 days of conservatory treatment and the patient was released from the hospital after 10 days.

One year after the initial event, the patient had a generalized seizure with loss of consciousness and tonic clonic movements. She was admitted to the hospital and started on carbamazepine 40mg/day. A MRI examination of the brain (Fig. 3) showed a well delimited, round-oval image of 32/8 mm, with bosselated contour, hemorrhagic parietal signal (hemosiderin deposits) and liquid internal signal, situated in the postcentral gyrus of the right parietal lobe. The aspect was suggestive for a chronic parenchymal hematoma. In proximity of this image, a small stellar vascular network was noticed after contrast administration. This venous angioma is drained to the superior sagittal sinus.

During hospitalization the carbamazepine doses were increased and the patient was released under treatment with Timonil retard 300mg, 3 tablets/day (900mg/day). Under this treatment, the patient was seizure-free for 2 years.

Three years after the initial event, in February 2007, the patient underwent stereotaxic radiosurgery (gamma knife) at the Timone clinic in Marseille, France. Periodic angiographic exams were recommended. Six months after the stereotaxic radiosurgery the patient underwent an angiographic examination (Fig. 4) which showed only mild improvement compared to the initial images.

One year after the gamma knife intervention the patient suffered two partial sensory seizures affecting the left hemibody but this was expected as it is a frequent secondary effect of the stereotaxic surgery. Throughout this period she had been under Timonil retard 900mg/day treatment.

In March 2009, two years after the gamma knife intervention and almost five years after the hemorrhage

from the AVM, another angiography was performed. It showed that the AVM was excluded from the cerebral circulatory system. (Fig. 5)

The patient was discharged with the recommendation of continuing the Timonil retard treatment and also returning to our clinic for regular check-ups in order to detect as early as possible any long-term complication that might occur after gamma-knife therapy, such as hemorrhage, delayed cyst formation, increase of seizure frequency, middle cerebral artery stenosis and increased white matter signal intensity on T2-weighted magnetic resonance imaging.

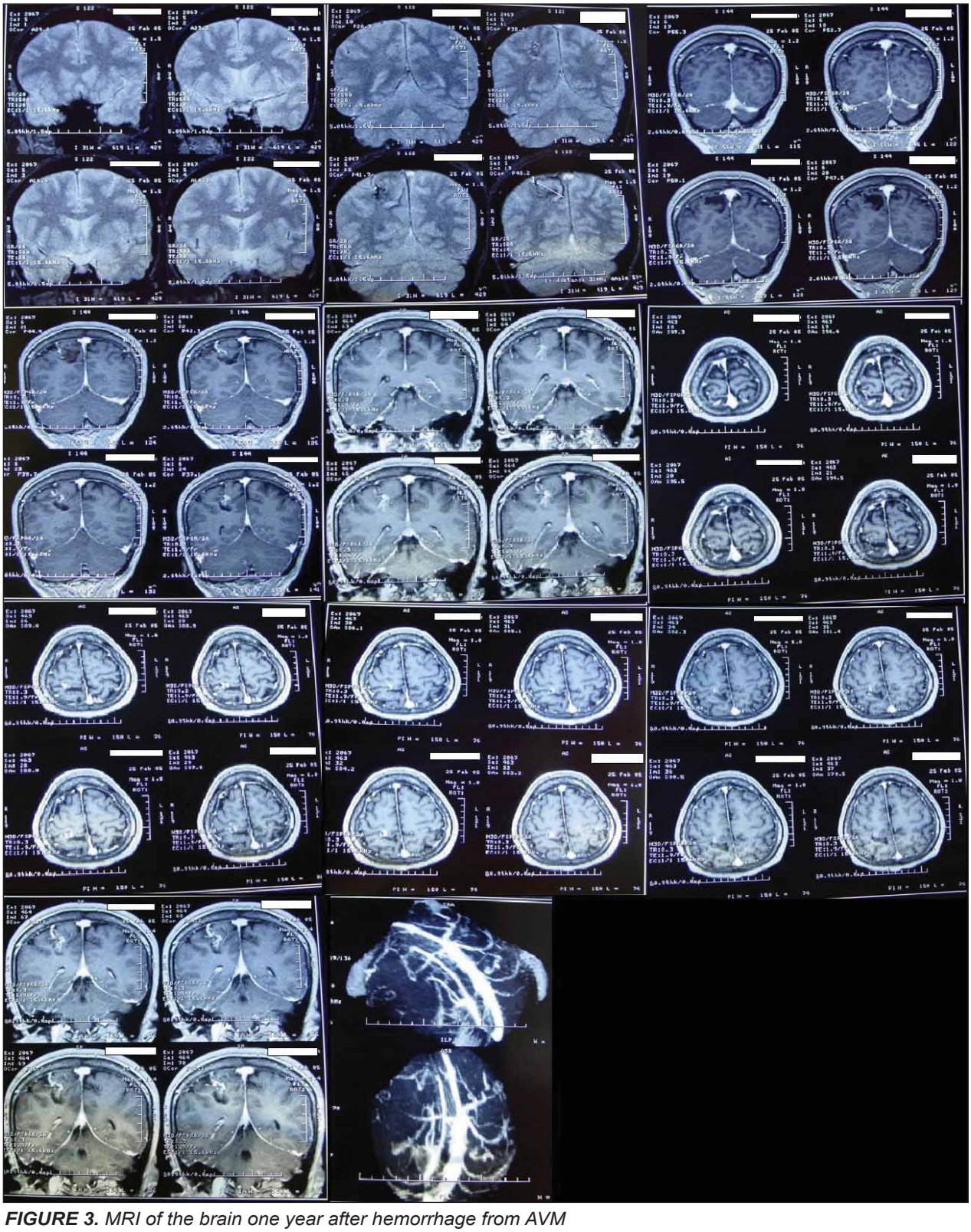

FIGURE 3. *MRI of the brain one year after hemorrhage from AVM*

FIGURE 4. *Angiography images three and a half years after hemorrhage, six months after gamma knife*

FIGURE 5. *Angiographic images two years after stereotaxic radiosugery*

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